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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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HELLER EHRMAN LLP 275 MIDDLEFIELD ROAD MENLO PARK, CA 94025-3506			MCKELVEY, TERRY ALAN	
			ART UNIT	PAPER NUMBER
			1636	

DATE MAILED: 10/04/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary

Application No.

10/006,116

Applicant(s)

BAKER ET AL.

Examiner

Terry A. McKelvey

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-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 05 July 2005.
- 2a) ☒ This action is **FINAL**. 2b) ☐ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 28-32 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 28-32 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
- ☐ Certified copies of the priority documents have been received.
 - ☐ Certified copies of the priority documents have been received in Application No. _____.
 - ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- | | |
|---|---|
| 1) <input type="checkbox"/> Notice of References Cited (PTO-892) | 4) <input type="checkbox"/> Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____ |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948) | 5) <input type="checkbox"/> Notice of Informal Patent Application (PTO-152) |
| 3) <input checked="" type="checkbox"/> Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08)
Paper No(s)/Mail Date <u>7/5/05</u> . | 6) <input type="checkbox"/> Other: _____ |

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DETAILED ACTION

All objections and rejections not repeated in the instant Action have been withdrawn due to applicant's response to the previous Action.

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Claim Rejections - 35 USC § 101 and 35 USC § 112, First Paragraph

Claims 28-32 are rejected under 35 U.S.C. 101 because the claimed invention is not supported by either a specific and substantial asserted utility or a well established utility. This rejection is maintained for reasons of record set forth in the paper filed 4/5/05. Applicants' arguments filed 7/5/05 have been fully considered but they are not deemed to be persuasive.

The claims are directed to antibodies that bind isolated polypeptides comprising SEQ ID NO:194, referred to as PRO1303 in the specification, and polypeptides that have 80% or higher amino acid sequence similarity. The specification does not disclose that PRO1303 has significant homology to other, prior art proteins. The instant specification does not disclose any additional information regarding PRO1303 such as subcellular

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location, timing of regulation during cellular differentiation, which hormones or transcription factors regulate PRO1303, and what physiological significance is possessed by PRO1303. The utility of anti-PRO1303 antibodies is linked to the utility of the PRO1303 polypeptide itself because the specification asserts that anti-PRO antibodies can be used in diagnostic assays for PRO, e.g., detecting its expression in specific cells, etc, and used for affinity purification of PRO. Therefore, the antibody has a utility if the protein it binds to, PRO1303, has utility.

The specification also generally asserts that all of the disclosed PRO polypeptides will be useful for a number of purposes; however, none of these asserted uses meet the three-pronged requirement of 35 USC 101 regarding utility, namely, that the asserted utility be credible, specific, and substantial. The asserted utilities will each be addressed in turn.

1. The PRO polypeptide can be used to isolate other polypeptides to which it binds. This asserted utility is not specific or substantial. Since the same can be done with any polypeptide, the asserted utility is not specific to the claimed PRO1303 polypeptide. Furthermore, since the specification does not disclose how PRO1303 or its binding partners can be used, significant further research would be required of the skilled

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artisan to identify and reasonably confirm a real world context of use because none are set forth simply by binding another protein.

2. The PRO polypeptide can be used as a molecular marker. This asserted utility is not specific since the same can be done with any polypeptide and thus is not specific to the PRO1303 polypeptide.

3. The PRO polypeptide can be used in tissue typing. The asserted utility is not specific or substantial. With the exception of a few housekeeping genes, all polypeptides have a tissue specific pattern of expression, and thus virtually any polypeptide can be used in tissue typing. Thus, the asserted utility is not specific to PRO1303.

4. The PRO polypeptide can be used in therapy. This asserted utility is not specific or substantial. Since a defect in any polypeptide is likely to cause a disease of some sort, every polypeptide is a target for drug development. Thus, the asserted utility is not specific to the claimed PRO1303 polypeptide. Furthermore, the specification does not disclose a nexus between any specific disease state and a change in amount or form of PRO1303. Significant further research would have to be conducted to identify such a nexus and to thus identify and

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confirm a real world context of use. Therefore, the asserted utility is not substantial.

5. The PRO polypeptide can be used to identify agonists or antagonists. Since the same can be done with any polypeptide, the asserted utility is not specific to the claimed PRO1303 polypeptide. Furthermore, since no activity has been assigned to PRO1303, the assay cannot be conducted until the specific biological activities of PRO1303 are determined empirically. Therefore, the asserted utility is also not substantial.

The specification also discloses that DNA encoding for PRO1303 tested positive in a gene amplification assay, with the DNA being amplified over 2-fold in primary lung tumors and colon tumors. The utilities asserted based upon this positive result are use as diagnostic markers for determining the presence of tumor cells in lung and/or colon tissue samples and utility in cancer therapy and screening for cancer therapeutics. Even though the DNA encoding PRO1303 has diagnostic utility based upon these results, the PRO1303 polypeptide (and thus the corresponding anti-PRO1303 antibody) does not for the following reasons. The increased copy number of DNA does not provide a readily apparent use for the polypeptide because there is no information regarding level of expression, activity, or role in cancer. Increased copy number of DNA in a cancer or transformed

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cell does not necessarily result in increased level of expression of the polypeptide, as shown by Konoka et al and Pennica et al. Konopka et al teach that: "Protein expression is not related to amplification of the abl gene but to variation in the level of bcr-abl mRNA produced from a single Ph1 template." (abstract). Pennica et al teach that: "In contrast, WISP-2 mapped to human chromosome 20q12-20q13 and its DNA was amplified, but RNA expression was reduced (2- to >30 fold) in 79% of the tumors." (abstract). These references thus show that even if amplification of a gene occurs in a tumor cell, it does not mean that the mRNA or protein expressed from the gene is also amplified and thus usable as a diagnostic marker for cancer. Since the protein is not necessarily overexpressed in cancer cells, then there is no substantial utility in using the protein for cancer therapy or screening for cancer therapeutics.

A substantial utility, by definition is a utility that defines "real world" use, and a utility that requires or constitutes carrying out further research to identify or reasonably confirm a "real world" context of use is not a substantial utility. In the instant case, the amplification of the DNA encoding PRO1303 is, at most, an interesting invitation for further research and confirmation as to whether PRO1303 protein itself is overexpressed or whether high PRO1303 protein

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expression plays an important role in cancer (and thus might be usable as a therapeutic target). This further research and experimentation, however, is part of the act of invention, and until it has been undertaken, the claimed invention is not considered to have utility based upon gene amplification in tumors.

The specification also discloses that PRO1303 tested positive as stimulators of glucose and/or FFA (free fatty acid) uptake. The asserted utility based upon this assay result is that the polypeptide would be expected to be useful for the therapeutic treatment of disorders where either stimulation or inhibition of glucose uptake by adipocytes would be beneficial for example, obesity, diabetes, or hyper- or hypo-insulinemia. The specification does not specifically assert how antibodies against PRO1303 would be used in any of the suggested treatments. First, the specification does not indicate which asserted utilities correspond specifically to glucose uptake stimulation as opposed to glucose uptake inhibition. Second, the specification does not indicate what, if any of the utilities set forth correspond to stimulation of FFA uptake. Third, the actual assay result is stimulation of glucose and/or FFA uptake, three very different activities (stimulation of glucose uptake only, stimulation of FFA uptake only, and

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stimulation of uptake of both). Would PRO1303 polypeptides be useful to treat hyper-insulinemia or would it be useful to treat hypo-insulinemia, two opposite conditions? Fourth, it is unclear how increasing uptake of FFA into adipocytes would treat obesity (or thus diabetes). Fabris et al teaches that in obesity, excessive energy storage as fat is mainly due to an imbalance between energy intake and expenditure, and the preferential channeling of excess calories as fat rather than protein or glycogen may play an important role in the development and maintenance of the disease. FFA-induced insulin resistance saves scarce glucose for central nervous system requirements, but this becomes counterproductive in obesity because it inhibits glucose utilization when there is no need to save it. Glucose and FFA might thus be channeled toward tissues (such as adipose tissue in which insulin sensitivity is maintained or even improved) (page 601, second column). Thus, increase of uptake of FFA and/or glucose into adipocytes does not appear to be a utility for treatment of obesity or diabetes.

Furthermore, the observed differences do not appear to be statistically significant and the cutoff points appear to be arbitrary and there is not obvious scientific basis for them. For example, Santomauro et al. (1999. Diabetes 48:1836-1841) teach that 56.5% decreases in FFA levels are statistically

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significant and correlated with physiological improvements, but it is not clear from either the prior art or the specification whether 50% decreases are useful (see Table 2 from Santomauro et al.). Note that 50% decreases in *plasma* insulin do appear to be significant, but it is not clear whether this is due to a doubling of insulin uptake by adipocytes or by other tissues, or whether it is due to changes in the amount of insulin production. Similarly, the observation that 56.5% decreases in *circulating* FFAs is significant and correlated with physiological improvements does not indicate that a doubling of uptake of FFAs *by adipocytes* will lead to the same decreases in FFAs. For example, doubling the amount of FFA uptake from 1% to 2% of total circulating FFAs would not be expected to lead to a 56% decrease in circulating FFA levels.

35 USC § 101 specifically requires that the invention must be useful in currently available form, which precludes any further experimentation to establish the utility of the claimed invention. Because the instant specification, as filed, fails to disclose a specific role of PRO1303 in glucose and/or FFA uptake in adipocytes, one would have reasons to conclude that the instant invention was not completed as filed, and, therefore, clearly lacks utility in currently available form.

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A substantial utility, *by definition*, is a utility that defines "real world" use, and a utility that requires or constitutes carrying out further research to identify or reasonably confirm a "real world" context of use is not a substantial utility. In the instant case, the mere fact that the protein encoded by the claimed nucleic acids was "positive" in two assays is at the most, an interesting invitation for further research, experimentation and confirmation as to whether the PRO1303 protein or its corresponding antibody is useful as a treatment for diabetes, obesity, hyper-insulinemia, or hypo-insulinemia. The further research and experimentation, however, is part of the act of invention, and until it has been undertaken, the claimed invention is not considered specific or substantial.

Claims 28-32 are also rejected under 35 U.S.C. 112, first paragraph. Specifically, since the claimed invention is not supported by either a credible, specific and substantial asserted utility or a well established utility for the reasons set forth above, one skilled in the art clearly would not know how to use the claimed invention.

Response to Amendment

The Polakis Declaration under 37 CFR 1.132 filed 6/29/05 is insufficient to overcome the rejections of claims 28-32 based upon not being supported by either a specific and substantial asserted utility or a well established utility and 35 U.S.C. 112, first paragraph as set forth in the last Office action because of the following reasons.

The Declaration is directed to arguments concerning strong correlation between changes of mRNA present in any cell type and the level of protein expressed from that RNA. This argument is not persuasive because the specification fails to teach that Pro 1303 mRNA expression is increased when the genomic DNA is amplified and thus this argument is not applicable in the instant case where only genomic DNA amplification of Pro 1303 DNA is taught. The part of the Declaration that is addressed in the applicant's arguments are addressed below in the following section.

The Goddard Declaration under 37 CFR 1.132 filed 6/29/05 is insufficient to overcome the rejections of claims 28-32 based upon not being supported by either a specific and substantial asserted utility or a well established utility and 35 U.S.C.

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112, first paragraph as set forth in the last Office action because of the following reasons.

The Declaration is directed to arguments concerning use of the TaqMan PCR technique to detect gene copy amplification. This argument is not persuasive because the applicability of this technique was not questioned in the rejections of record and thus the utility of gene amplification for the instant Pro 1303 antibody with regard to detection of gene copy number amplification for diagnostic purposes is accepted.

The Ashkenazi Declaration under 37 CFR 1.132 filed 6/29/05 is insufficient to overcome the rejections of claims 28-32 based upon not being supported by either a specific and substantial asserted utility or a well established utility and 35 U.S.C. 112, first paragraph as set forth in the last Office action because of the following reasons.

The Declaration argues that even when amplification of a cancer marker gene does not result in significant over-expression of the corresponding gene product, this very absence of gene product over-expression still provides significant information for cancer diagnosis and treatment, such as if the gene product is overexpressed in some tumor types, but not others, then parallel monitoring of gene amplification and gene

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product over-expression enables more accurate tumor classification and hence better determination of suitable therapy. If the gene is amplified but the corresponding gene product is not overexpressed, the clinician accordingly will decide not to treat a patient with agents that target that gene product. This argument is not persuasive because in the instant case, Pro1303, there is no description in the specification whether the Pro1303 protein expression is always, sometimes, or never overexpressed in tumors where the Pro1303 gene has been amplified, which is indicative of cancer. Therefore, there is no nexus shown between the parallel monitoring and tumor classification/determination of suitable therapy. And, because there is no description of an agent in the art or specification which targets Pro1303, then monitoring Pro1303 levels in tumors does not have utility in deciding how to treat the patient.

Response to Arguments

Regarding the arguments concerning specifically gene amplification alone, these arguments are not persuasive in overcoming the rejections because gene amplification of Pro1303 DNA and the Pro1303 DNA's utility is not being questioned in the instant rejections.

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The applicant argues that increased copy number does not necessarily result in increased polypeptide expression, but the Examiner has not shown whether it is more likely than not that such correlation does not exist, and that it is a working hypothesis among those skilled in the art that if a gene is amplified in cancer, the encoded protein is likely to be expressed at an elevated level. These arguments are not persuasive because the standard is that it is more likely than not that one of ordinary skill in the art would doubt the truth of the statement of utility. One of ordinary skill in the art is a Ph.D. scientist with at least a few years experience postgraduate. From the uncertainty in the cited art concerning the correlation between gene amplification and gene product overexpression, one of ordinary skill in the art would doubt the alleged utility because in the absence of information showing the correlation, one would doubt that the correlation exists. In other words, one of ordinary skill in the art would not assume that the alleged utility is correct, just based upon an assertion of the utility, because one of ordinary skill in the art would recognize the uncertainty concerning any correlation and thus one of ordinary skill would doubt the utility until the alleged correlation is empirically determined. The applicant's

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statement concerning the working hypothesis was not supported and thus is not persuasive.

The applicant argues that based upon some cited references, such as one which teaches that 62% of highly amplified genes show moderately or highly elevated expression, and that on average, a 2-fold change in copy number is associated with a corresponding 1.5-fold change in RNA levels. This argument is not persuasive because the value of 62% very much shows the uncertainty in the art concerning the correlation of DNA copy number and RNA expression itself. The average correlation is not persuasive because a particular amplified gene/mRNA can be very different from the average and thus must be empirically determined, which was not done for Pro1303. The applicant/Polakis Declaration argues that there is 80% of the time a correlation between mRNA and corresponding protein levels. This argument is not persuasive because this figure shows the uncertainty in the art whether there is any correlation between overexpressed RNA and overexpressed protein. Additionally, since the lack of utility is based upon the lack of utility of the protein as a diagnostic marker for cancer, the proper uncertainty is determined by 62% of the correlation between DNA/RNA multiplied by 80% of the correlation between RNA/protein to arrive at a correlation between DNA/protein of

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less than 50%. This uncertainty is even that much stronger, and thus one of ordinary skill in the art would certainly be more likely than not to doubt the truth of the statement of utility.

The applicant's arguments concerning the Ashkenazi Declaration are addressed in the section above. The applicant's arguments concerning HER-2/neu gene are not persuasive because this gene has a known partial correlation between gene amplification and protein overexpression and uses it. This is not analogous to the instant Pro1303 situation where even a partial correlation is unknown.

The applicant argues that it was well known in the art that increasing glucose uptake by adipocyte cells is a hallmark of a number of therapeutically effective agents, such as to treat diabetes. However, the specification does not actually describe whether Pro1303 actually increases glucose uptake because the assay results were set forth as being positive for glucose and/or FFA uptake. If PRO1303 was positive for only FFA uptake, then the arguments concerning utility based upon glucose uptake are not persuasive. The applicant argues that FFA uptake is closely related to glucose uptake. However, this argument is not persuasive because the applicant fails to show that uptake of FFA is a hallmark of therapeutically effective agents to treat the indicated diseases. The remaining arguments concern

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FFA levels and correlation with disease treatment. These arguments are not persuasive because there is still no indication that compounds in the art which increase of FFA alone (not increasing glucose uptake) are effective for the treatment of any particular disease.

Therefore, in light of all available evidence, including the rejection set forth previously and repeated above, the applicant's arguments and declarations, and the instant arguments set forth, the claims are not supported by either a specific and substantial asserted utility or a well established utility and thus one skilled in the art clearly would not know how to use the claimed invention. Therefore, the rejections are properly maintained.

Claim Rejections - 35 USC § 102

Claims 28-32 are rejected under 35 U.S.C. 102(e) as being anticipated by Ni et al (U.S. Patent No. 6,566,498 B1). This rejection is maintained for reasons of record set forth in the paper filed 4/5/05. Applicants' arguments filed 7/5/05 have been fully considered but they are not deemed to be persuasive.

Ni et al teach an isolated human secreted polypeptide consisting of SEQ ID NO:6, which has two regions of 100% identity with a polypeptide consisting of SEQ ID NO:194, one 62

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amino acids long (the first 62 amino acids) and one about 93 amino acids long (at the C-terminus). These large stretches of perfect identity between the two proteins only at each terminus of the proteins would appear to indicate that the two proteins are likely splice-variants of each other and thus are very likely to have many exterior-exposed epitope domains in common. See the attached sequence comparison. Also, because antigenic epitopes can be as low as 7 amino acids and preferably between 15 and 30 amino acids (Column 20 of Ni et al), many of the antibodies taught by the reference which are directed against the protein of SEQ ID NO:6 would strongly cross-react with and specifically bind to the polypeptide of SEQ ID NO:194.

"Specifically binds" is interpreted in the claims as broad as is reasonable in the art, which encompasses antibody binding to a protein with a high affinity, of a level comparable with proteins having the identical epitope. The many stretches of 15 to 30 amino acids in common shows that many epitopes that would generate antibodies that specifically bind are in common between the two proteins. Monoclonal and polyclonal antibodies are taught, as are antibody fragments, labeled antibodies, and humanized antibodies (columns 20-21 and 26).

Response to Arguments

The applicant argues that "specific binding" means that an antibody binds to a unique epitope within a target sequence (without cross-reacting with another epitope, including those found in the sequence disclosed in Ni et al). It is argued that in view of this the Examiner errs in assuming that antibodies claimed in the present application would display significant binding to the polypeptide of Ni et al. The applicant argues that there exists specific epitopes in SEQ ID NO:194 and that one of ordinary skill in the art would readily understand that what is meant by antibodies that specifically bind to SEQ ID NO:194 (and not, for example, to the polypeptide of Ni et al).

This argument is not persuasive for the following reasons. First, it should be noted that even just one antibody taught by the reference which specifically binds to the Ni et al protein and specifically binds to the polypeptide of SEQ ID NO:194 would anticipate the claimed invention. It is the Examiner's position that based upon the several large regions of perfect sequence identity, not only is there one such antibody, but a large plurality of such antibodies taught by the reference which would meet the claim limitations of specifically binding to the polypeptide of SEQ ID NO:194.

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Applicant's definition of "specific binding" is too restrictive and does not interpret the phrase as broadly as is reasonable. "Specifically binds" is interpreted in the claims as broad as is reasonable in the art, which encompasses antibody binding to a protein with a high affinity, of a level comparable with proteins having the identical epitope. High affinity binding occurs when there is a sufficient number of non-covalent bonds formed between the antibody and the epitope to which it binds which results in a relatively strong and stable association between the antibody and the epitope (and thus the protein having that epitope). It is not defined relative to whether or not the antibody specifically binds to the same epitope present in a different protein, especially since it is unclear what would constitute a different protein in this context. If an antibody binds with high affinity to the claimed protein and binds with high affinity to a non-SEQ ID NO:194 protein such as SEQ ID NO:194 having one conservative substitution, does it constitute an antibody which specifically binds to the polypeptide of SEQ ID NO:194? If it doesn't then no antibody would specifically bind to the polypeptide of SEQ ID NO:194 because every possible antibody that binds to any particular epitope in SEQ ID NO:194 would also bind to that epitope when present in another protein that is not SEQ ID

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NO:194. If it is encompassed by the definition, then how different would the protein have to be before it is no longer encompassed? The Ni et al protein is clearly related to SEQ ID NO:194 because of its large regions of sequence identity and thus could be seen as SEQ ID NO:194 having a certain number of differences due to mutation. This argument thus shows that applicant's arguments concerning the definition of "specifically binds" is not only incorrect, but also untenable.

The applicant argues that an antibody generally recognizes only a small region on the surface of a large molecule and that most antibodies against intact, fully folded proteins recognize discontinuous epitopes, and that for this reason, the binding sites for the claimed antibodies cannot be simply predicted based on the linear sequence homology between the amino acid sequence of the present invention and that of Ni.

This argument is not persuasive for the following reasons.

First, the regions of perfect sequence identity between the Ni protein and SEQ ID NO:194 are large enough to constitute folded domains in which there are epitopes which are formed from amino acids on the surface of the folded domains which are all present in the region of perfect identity and thus would bind to that epitope present in both proteins.

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Second, the claims are not drawn to an antibody that specifically binds to the polypeptide of SEQ ID NO:194 wherein the polypeptide is fully folded. The claims encompass antibodies that specifically bind to any form of the polypeptide of SEQ ID NO:194, including denatured, but intact forms of the polypeptide of SEQ ID NO:194. Such antibodies are much more likely to have epitopes that are a small stretch of contiguous amino acids or almost contiguous amino acids. Thus, the large regions of identity between the two proteins would certainly have such epitopes in common and thus antibodies which specifically bind to such epitopes would specifically bind to both the Ni et al protein and SEQ ID NO:194.

Finally, the applicant indicates that it was well known in the art how to make and use antibodies. This argument is not persuasive in overcoming the instant rejection because it is drawn to enablement, not whether the cited prior art anticipates the claimed invention.

Conclusion

No claims are allowed.

THIS ACTION IS MADE FINAL. Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

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A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

Certain papers related to this application may be submitted to Art Unit 1636 by facsimile transmission. The faxing of such papers must conform with the notices published in the Official Gazette, 1156 OG 61 (November 16, 1993) and 1157 OG 94 (December 28, 1993) (see 37 C.F.R. § 1.6(d)). The official fax telephone number for the Group is 571-273-8300. NOTE: If Applicant does submit a paper by fax, the original signed copy should be retained by applicant or applicant's representative. NO DUPLICATE COPIES SHOULD BE SUBMITTED so as to avoid the processing of duplicate papers in the Office.

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Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

Patent applicants with problems or questions regarding electronic images that can be viewed in the Patent Application Information Retrieval system (PAIR) can now contact the USPTO's Patent Electronic Business Center (Patent EBC) for assistance. Representatives are available to answer your questions daily from 6 am to midnight (EST). The toll free number is (866) 217-9197. When calling please have your application serial or patent number, the type of document you are having an image problem with, the number of pages and the specific nature of the problem. The Patent Electronic Business Center will notify applicants of the resolution of the problem within 5-7 business days. Applicants can also check PAIR to confirm that the problem has been corrected. The USPTO's Patent Electronic Business Center is a complete service center supporting all patent business on the Internet. The USPTO's PAIR system provides Internet-based access to patent application status and history information. It also enables applicants to view the scanned images of their own application file folder(s) as well as general patent information available to the public.

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Any inquiry concerning rejections or objections in this communication or earlier communications from the examiner should be directed to Terry A. McKelvey whose telephone number is (571) 272-0775. The examiner can normally be reached on Monday through Friday, except for Wednesdays, from about 7:30 AM to about 6:00 PM. A phone message left at this number will be responded to as soon as possible (i.e., shortly after the examiner returns to his office).

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Dr. Remy Yucel can be reached at (571) 272-0781.



Terry A. McKelvey, Ph.D.
Primary Examiner
Art Unit 1636

September 29, 2005